# 53 | Muscle Cramps

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## Definition

Muscle *cramp* denotes an episodic, involuntary, painful contraction of a muscle. Muscle *spasm* is a more encompassing term referring to any involuntary muscle contraction.

## Technique

Muscle cramps are ubiquitous yet tend to be little more than a benign nuisance to the sufferer. On occasion, though, the physician encounters either patients who complain of "cramps" or might want to inquire as to their presence while evaluating a related symptom.

There is nothing unique in taking a history of cramps, the aims being both to characterize the complaint fully and to extract clues that might reveal its cause. This can be accomplished by having the patient describe the symptom, then asking directed questions as necessary to elaborate information as to the quality, location, duration, precipitating and relieving maneuvers, course of the symptom, and associated features.

Many patients who complain of "cramps" actually have some other related phenomenon. What characteristics, then, can be helpful in making the correct diagnosis?

- *Quality.* It is useful to find out about onset (abrupt or slow?), sensation (painful? stiff?), and appearance (if an abnormal posture is described, have the patient demonstrate it). With true cramps, the onset is sudden, the muscle feels taut and painful, and usually it is visibly and palpably knotted (although this may not be readily apparent if only part of the muscle is involved).
- *Location.* Determine if muscles are involved singly or in groups, if certain muscles are consistently involved, and if the cramps remain limited or spread. While ordinary cramps often affect the leg (especially calf) muscles, other locations may point to a specific syndrome such as carpopedal spasms in tetany or unilateral facial involvement in hemifacial spasms.
- *Duration.* Ordinary cramps last from seconds to several minutes if severe. Fleeting twitches or prolonged contractions suggest another type of disorder.
- *Precipitating or relieving maneuvers.* Attempt to uncover any relation to exercise; specifically, find out not only if occurrence is at rest or during or after exercise but also the duration and intensity of exercise that may initiate cramps. Moreover, clarify if the cramps appear only sporadically or can be predictably expected at a certain level of exertion. One may also want to ask about other potential triggers such as movement, sensory or emotional stimuli, hyperventilation, cold, or fasting. Assess if anything alleviates the cramps.
- *Course of symptoms.* A relatively recent onset suggests the possibility of an acquired intercurrent illness, whereas affliction since youth may point to an inherited disorder.

It is important to recognize several entities that may be confused with cramps. With disorders of muscle relaxation, as in myotonia and hypothyroidism, the patient may perceive an uncomfortable but painless muscle tightness or stiffness, often made worse in the cold. One can get a clue to myotonia if the patient admits to difficulty releasing a grip (be it a handshake, doorknob, or cooking pan) that improves with repeated contractions. Other rare ailments can produce a sensation of stiffness via various mechanisms (see Table 53.1).

Focal dystonias share certain similarities and may easily be mistaken for cramps. Dystonia refers to a movement disorder manifest by the assumption of involuntary abnormal postures. Patients may relate spasmodic, even painful, bending or twisting movements of an extremity, neck, face or trunk that remain in a persistent attitude (e.g., overextended/overflexed posture of hand, inversion of foot, pulling of the head to one side). Sometimes the spasms occur in relation to and interfere with a specific skilled motor act, as in so-called writer's cramp. The slower onset, inappropriate contraction of many opposing muscles, and unusual postures evoked help to distinguish dystonia.

Far and away a more common patient complaint is that of a "crampy" feeling, by which is meant an aching discomfort in the muscles without any visible or palpable hardening. These myalgias are common in childhood. They can also be seen in a variety of neuromuscular and systemic illnesses, as well as in the context of generalized pervasive fatigue without objective muscle weakness in depressed patients.

It should be emphasized, though, that the above conditions, along with other abnormal muscle contractions, may occur in conjunction with actual muscle cramps. Patients as historians can be "lumpers" or "splitters"—they may complain of cramps as one of many symptoms, or include a number of different phenomena under the rubric of "cramps."

Once one has fully characterized the symptoms, the next step is to probe for associated features that may disclose an etiology. Appropriate queries might include: Is there any weakness, numbness, or paresthesias present that may indicate an underlying neuromuscular disorder? Has muscle twitching been noted? Fasciculations (spontaneous visible muscle twitches due to contraction of a motor unit) can be seen in several neuropathic disorders associated with cramping. They can also appear as a benign condition with muscle cramps, particularly in people with large muscles.

Do bouts of myoglobinuria occur? This sign, reflecting muscle fiber breakdown, can be ascertained by asking if the

**Table 53.1**
The Cramp History

| Cramps/spasms | Principal complaint | Provoked by: | Typical distribution | Associated complaints |
|---|---|---|---|---|
| Ordinary cramps | Brief, painful cramps | Trivial movement, exercise, sleep | Legs | See Table 53.2 |
| Glycogen storage myopathies (e.g., myophosphorylase, phosphofructokinase deficiency) | Brief or prolonged (rare), painful cramps | Vigorous exercise | Exercising muscles | Myalgias, myoglobinuria |
| Carnitine palmityl transferase deficiency | Brief or prolonged (rare), painful cramps | Prolonged exercise | Exercising muscles | Myalgias, myoglobinuria |
| Tetany | Painless, carpopedal spasms | Hyperventilation | Hands, feet | Paresthesias |
| Hemifacial spasms | Intermittent twitches or prolonged spasms of face | Facial movement | Facial muscles | |
| Flexor spasms | Involuntary flexion of legs | Cutaneous or visceral stimuli | Thigh, knee, foot | Stiffness, autonomic symptoms |
| *Slow relaxation* | | | | |
| Myotonia (myotonic dystrophy, myotonia congenita, paramyotonia, chondrodystrophic myotonia) | Painless difficulty releasing grip, etc. | Strong contractions, cold, percussion | Limbs, face | Related to underlying disease |
| Hypothyroidism | Muscle "tightness," slow contraction/relaxation | Activity, rest, cold | Limbs, face | May or may not have cramps, proximal weakness, myalgias |
| *Stiffness* | | | | |
| Neuromyotonia | Persistent, painless stiffness | Exercise | Diffuse | Delayed relaxation, myokymia, cramps |
| Tetanus | Persistent stiffness, painful cramps | Movement, sensory or emotional stimuli | Diffuse (can be localized); trismus frequent | |
| Stiff man syndrome | Persistent stiffness, painful cramps | Movement, sensory or emotional stimuli | Diffuse (trismus not prominent) | |
| Strychnine poisoning | Persistent stiffness, painful cramps | Movement, sensory or emotional stimuli | Diffuse | |
| Black widow spider bite | Persistent stiffness, painful cramps | Spontaneous | Abdomen, trunk | Myalgias, paresthesias, autonomic and CNS symptoms |

urine ever becomes cola colored. Be careful—patients frequently say their urine becomes dark (concentrated) but rarely admit that it actually turns brownish.

Are there any concurrent illnesses or medications? Spontaneous muscle cramps are associated with a number of conditions and medications (see Table 53.2).

Is there a family history of cramps?

**Table 53.2**
Clinical Settings of Muscle Cramps

*Neuromuscular disorders*
Motor neuron disease (may be presenting symptom)
Nerve root irritation
Peripheral neuropathies

*Medical disorders*
Sodium and water loss (e.g., diarrhea)
Hypothyroidism
Uremia
Hemodialysis
Pregnancy

*Drugs*
Acetylcholinesterase inhibitors
Diuretics
Clofibrate

*Healthy individuals*
Exhaustive exercise (during or after)
Benign fasciculations—large muscles
Familial tendency
"Idiopathic" nocturnal leg cramps

## Basic Science

Normal voluntary motor activity depends on the functional integrity of not only the motor unit (comprising a lower motor neuron and the muscle fibers it innervates) but also the descending and spinal excitatory and inhibitory circuits that influence it. Cramps may result from malfunction at various points in the pathway.

Muscle fibers contract in response to impulses generated by the lower motor neurons. With the arrival of these impulses, or action potentials (AP), at the presynaptic nerve

terminals, the neurotransmitter acetylcholine is released. If there are sufficient amounts of acetylcholine to activate the postsynaptic receptors, a muscle AP is produced. This AP is then transmitted to the interior of the muscle fiber where it liberates calcium stored in the sarcoplasmic reticulum (a system of tubules that surround the contractile filaments, actin and myosin). The calcium binds to a regulatory protein, permitting the chemical interaction of actin and myosin, which results in muscle shortening. The energy for contraction is supplied by hydrolysis of adenosine triphosphate (ATP). Muscle relaxation depends on active reuptake of calcium by the sarcoplasmic reticulum. This reuptake, too, is an energy-dependent process requiring ATP. Thus, if ATP is lacking, the muscle remains shortened even in the absence of continued action potentials. This is known as *contracture*.

If, alternatively, reuptake occurs but a second AP arrives before the muscle has relaxed, the contraction will be continued. At motor neuron firing frequencies of 10 to 20 Hz, the twitches fuse into a sustained contraction. The activity of the motor neuron is modulated by spinal interneurons, which can, via presynaptic or postsynaptic inhibition, decrease motor neuron excitability.

The energy for muscle contraction can be produced by either carbohydrate or fatty acid metabolism. Oversimply put, resting muscle predominantly utilizes lipid. During intense anaerobic exercise, additional ATP is generated through glycogen breakdown. With more prolonged mild to moderate activity, however, fatty acids again provide the principal fuel.

From this scheme, one could postulate a number of mechanisms for muscle cramps. There may be impairment of normal descending or spinal inhibitory pathways with subsequent motor neuron overactivity. Or there could be hyperirritability of motor nerves anywhere from the cell body to the terminal muscular branches. At the level of the muscle fiber, a deficiency of ATP might prevent calcium reuptake into the sarcoplasmic reticulum, leaving the fiber in a shortened state. Indeed, examples of every mechanism are thought to exist and are described in the next section.

## Clinical Significance

As mentioned previously, cramps can be due to a variety of causes. Some cramps remain benign and mysterious; others are of a more definite nature and respond to specific therapy.

*Ordinary muscle cramps* are characterized by a painfully hard, palpable contraction of explosive onset, at times preceded or followed by brief twitches, which usually involves one muscle at a time. They may be precipitated by volitional movement of trivial extent or more forceful contraction in a muscle already shortened (as may occur during or after exercise). The cramps can be terminated by passive stretching of the muscle or activation of its antagonist. In severe cramps, the muscle may remain sore and tender for days.

While leg cramps are fairly common in the elderly, and hardly unusual in otherwise healthy, athletic individuals, they appear more frequently in certain clinical settings (see Table 53.1). The mechanism of such a relatively familiar malady has remained obscure, although a popular theory suggests that the intramuscular nerve branches are for some reason rendered hyperexcitable.

Clinically similar but on a different pathophysiologic basis are the cramps, or more correctly, contractures of muscle associated with various *metabolic myopathies*. In several inherited disorders of glycogen metabolism, such as myophosphorylase (McArdle's) or phosphofructokinase (Tauri's) deficiency, patients classically experience myalgias and painful, hard muscle cramps only during vigorous exercise. Occasionally, an episode of cramps will be followed by myoglobinuria, which, if excessive, could induce acute renal insufficiency. Conversely, patients deficient in carnitine palmityl transferase, an enzyme critical for fatty acid oxidation, get cramps only during prolonged exercise, especially if in the fasted state, on a low-carbohydrate diet, or in cold weather. The proposed physiologic thread that ties these conditions together is acute depletion of ATP during exercise, disrupting calcium reuptake into the sarcoplasmic reticulum and preventing muscle relaxation.

*Tetany* is a cramplike syndrome consisting of painless spasms, most notable for their carpopedal distribution but potentially involving other muscles. The hand typically assumes a posture of tonic adduction of thumb and fingers, flexion of metacarpophalangeal joints, with extension of interphalangeal joints. The toe becomes plantar flexed and the ankle inverted. The spasms, commonly precipitated by hyperventilation, are preceded by tingling paresthesias of the mouth, fingers, and toes, a discriminative feature from dystonia. Tetany is seen in states that cause alkalosis or reduction in ionized calcium and magnesium, which appear to render the peripheral nerves hyperirritable.

A more focal disorder of peripheral nerve function is *hemifacial spasm*. Here, potentially any facial movement (e.g., blinking) may provoke either fine twitching or prolonged gross spasms of facial muscles. It is believed that most cases are attributable to compression of the facial nerve in the posterior fossa.

*Tetanus*, in contrast, is due to the suppression of spinal inhibitory neurons by an exotoxin of the bacterium *Clostridium tetani*. Clinically, involuntary spasms triggered by movement, sensory or emotional stimuli are superimposed on a state of virtually continuous stiffness. The symptoms may begin in the vicinity of a wound and remain localized or, more often, become generalized. Even in the localized form, one may note mild trismus due to the susceptibility of the masseter innervation. Persistent muscular rigidity and painful cramps can also occur with strychnine poisoning, black widow spider bites, and the rare "stiff man" syndrome.

Patients with prominent spasticity due to spinal cord lesions frequently complain of stiffness, muscle cramps, and spasms. Presumably, damage to the descending motor pathways disrupts the normal suprasegmental inhibitory influences on the spinal motor neurons, allowing afferent sensory impulses to evoke unbridled reflex activity. As a result, *flexor spasms* of the thigh, knee, and foot can be set off by cutaneous or visceral (e.g., distended bladder) stimuli. Being reflexogenic in nature, they are at times accompanied by such autonomic responses as sweating, piloerection, or incontinence.

Other involuntary muscle hyperactivity states, including several rare familial syndromes, have been described and are reviewed elsewhere (Layzer, 1971, 1979; Sumi et al, 1984).

## References

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